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Effect Of Diabetes Mellitus On Trace Elements Concentrations In Egyptian Diabetic Patients

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Abstract

Introduction: Diabetes mellitus is considered a primary cause of diabetic complications and is associated with impaired trace elements.

Aim of the Work: study effect of diabetes mellitus on Ttrace elements levels.

Material and Method: The study was conducted on 90 persons (30 Control, 30 Type I, 30 Ttype II). Diabetic patients with complications were excluded. Serum (or plasma) levels of Zn, Cu, Mg, Mn, Cr and Se were measured. Blood samples were collected in metal-free tubes from patients and controls after overnight fast. Weight and height were recorded.

Result and Discussion: Zn, Mg, Se level in type I diabetics showed no significant difference. Cu and Mn concentrations were significantly higher in both type I, II than controls. Zn, Se and Mg levels were significantly decreased in type II than control. Cr concentration were significantly lower in both type I, II than control. Mn is the most influential variable among the tested variables.

Conclusion: Diabetes can alter copper, zinc, magnesium, manganese, chromium and selenium levels and it needs further investigation.

Keywords: Trace Elements, Diabetes Mellitus, Zinc, Copper, Manganese, Magnesium, Chromium & Selenium.

Introduction

Diabetes mellitus is associated with impaired trace elements. Alterations in serum (or plasma) concentrations of several trace elements have been reported to occur in both type 1 and II diabetes mellitus. These micronutrients are suspected to have a role in the pathogenesis and progression of the disease (Zargar *et al.,* 2002). Zinc (Zn) is an essential mineral that is required for various cellular functions. Zn help in insulin absorption (Chausmer, 1998). Its abnormal metabolism is related to certain disorders such as diabetic complications. Levine *et al.* (1983) observed that the genetically insulin-resistant, diabetic mice demonstrate tissue zn deficiency. Zinc has an important role in the control of carbohydrate metabolism, and diabetic patients are at risk for zinc deficiency. Molecular and cellular studies have demonstrated several roles for zinc in insulin production and the consequent actions of insulin on metabolism. Copper (Cu) deficiency results in swelling and subsequent disruption of mitochondria of metabolically active tissues like hepatocytes and pancreatic acinar cells. Magnesium (Mg), the second most abundant intracellular cation, is indispensable to cellular metabolism. Mg is known to play an important role in carbohydrate metabolism and its imbalance has been implicated in diabetes mellitus both as a cause and a consequence (Paolisso *et al.,* 1990). Direct association of trace elements in relation to human disease has been observed in many research studies. The role of trace elements in some of the metabolic dysfunctions is not clear. Trace elements are uniquely required for growth and maintenance of life and health. Lack or an inadequate supply of such nutrients produces a functional impairment or can result in disease. The clinical significance and evaluation of trace elements such as Zn, Cu, Mn and Mg in regard to different diseases including diabetes mellitus remain conflicting as well as controversial and many questions still remain unanswered. Magnesium and zinc are nutritional

minerals that play crucial roles in the regulation of carbohydrate and lipid metabolism (Anetor *et al.*, 2002).

Aim of the work

The studies of zinc (Zn), copper (Cu), magnesium (Mg), manganese (Mn) chromium (Cr) and selenium (Se) levels in diabetic patients have led to contradictory findings. In the present study we tried to study the effect of diabetes mellitus on trace elements levels (Zn, Cu, Mg, Mn, Cr & Se) in serum of egyptian diabetic patients.

Material and Methods

The study consisted of analysis of serum levels of Zn, Cu, Mg, Mn, Cr and Se in egyptian patients with diabetes mellitus. The experimental group(s) consisted of 60 patients (30 Type I, 30 Type II). The control group consisted of 30 healthy volunteers with no general complications and receiving no medication. They were matched for age and sex. Age range: 30-45 years. Patients were selected from governmental and nongovernmental organization in Cairo who attended the diabetic clinics and were taking insulin (IDDM); pills or pills and insulin (NIDDM). The diagnosis of type 1 & 2 diabetes mellitus is based on the criteria of the expert committee on the diagnosis of diabetes Mellitus (2000). Diabetic patients with complications were excluded according to their medical history. None of the study subjects had taken any vitamin or mineral supplements. Blood samples were collected in metal-free tubes from patients and controls after an overnight fast. Serum was collected by centrifugation and stored in metal-free tubes at –70 °c. Minerals were measured in the ash using atomic absorption, unicam 929 (AOAC 1984). Glucose was determined according to Trinder (1969).

Preparation Of The Samples (Serum)

Samples for: Cu, Zn, Mn and Mg were prepared according to Price (1972), cr was prepared according to Victor *et al.* (1994), se was prepared according to Lioyd *et al.* (1982).

Anthropometrics measurements

Weight and height were measured in indoor clothing without shoes and body mass index (BMI) was calculated where BMI=wt (kg)/ht (m^2).

Statistical Analysis

Data are expressed as mean ±se. Data were assessed by t-test (Avram 1964; Steel and Torrie 1969). The correlation coefficients were determined by pearson's simple linear regression analysis (Steel and Torrie 1969). Statistical significance was accepted at $P < 0.05$. Multiple linear regressions were done using SPSS 13 package.

Results

Data of table (1) & figure (1) shows the characteristic of diabetic and control subjects. Data of table (1) reveal that no significant difference was found between the mean ages, and bmi of diabetic patients (type I & II) and control persons or between type I & II. Data of table (1) reveal that blood glucose level of diabetic patients (type I & II) are significantly higher than control group.

Data of table (2,4) & figure (1) shows the concentration of Zn, Cu level and cu/zn ratio of diabetic and control subjects. Data of table (2) reveal that Zn level of diabetic patients (IDDM) shows no significant change when compared with the control group, while niddm shows a significant decrease when compared with control or iddm group ($P < 0.01$ & 0.01). Data of table (2) reveal that Cu level, Cu/Zn ratio of diabetic patients (IDDM, NIDDM) shows significant increase when compared with the control group, and the increase was higher in niddm. Cu shows insignificant change when compared with control group, while cu/zn ratio showed a significant change when compared with iddm group ($P < 0.01$).

Data of table (3,4) & figure (1) shows the concentration of mg, mn, se and cr of diabetic and control subjects. Data of table (3) reveal that Mg & Se level of diabetic patients (IDDM) shows insignificant change when compared with the control group, while niddm shows a significant decrease when compared with the control

group (P<0.01). Data of table (3) reveal that mn level of diabetic patients (iddm, niddm) shows significant increase (P<0.01) when compared with the control group, and the increase was higher in niddm. Cr (iddm, NIDDM) shows significant decrease when compared with the control group (P<0.01). Mg & Mn showed insignificant difference when comparing iddm with niddm group, while Se & Cr showed the opposite i.e. significant difference was observed when comparing iddm with niddm group (IDDM > NIDDM group).

Correlation Coefficient

No significant correlations were found between most variables with some exception. Correlation was found between Cr and Se (r= -0.505; P<0.004 for control group; r=-0.90; P<0.000 for diabetic type II group). No significant correlation between glucose and zn was found which agree with (Anetor *et al.*, 2002). No significant correlation between glucose and mg was found which agree with Anetor *et al.* (2002).

Multiple Linear Regression Analysis

In multiple linear regression analysis using stepwise analysis model, the independent variable (s) were: weight, height, BMI, Cu, Zn, Mg, Mn, Se, Cr the dependent variable was glucose. Table (5) shows that mn is the most influential variables among the tested variables.

Table (1): Characteristics of the diabetic patients

		Age (Y)			BMI (kg/m^2)			Blood Glucose		
		Control	Type i (d_1)	Type II (D_2)	Control	Type I (D_1)	Type II (D_2)	Control	TYPE I (D_1)	TYPE II (D_2)
Mean±SE		38.84±1.30	38.43±1.17	37.75±1.26	23.64±0.47	23.47±0.51	23.38±0.57	88.82±3.16	213.32±6.36	229.15±7.04
D vs C	P≤		NS	NS		NS	NS		0.01	0.01
D_1 vs D_2	P≤			NS			NS			NS

Ns: Non significant, Very highly significant, P<0.001

Table (2): Zn, Cu level and Cu/Zn ratio of the diabetic patients

	Control	Type I	Type II	Control	Type I	Type II	Control	Type I	Type II
	Zn			Cu			Cu/Zn		
Mean	0.92	0.94	0.78	1.02	1.20	1.32	1.13	1.30	1.77
±SE	0.02	0.02	0.03	0.05	0.05	0.05	0.06	0.07	0.11
%		+ 2.58	- 14.63		+17.69	+ 28.50		+14.99	+ 56.90
P< D vs C		Ns	0.01		0.01	0.01		NS	0.01
D_1 vs D_2			0.01			NS			0.01

Ns: Non significant, Highly significant (P<0.01),

Table (3): Mg, Mn, Se and Cr level of the diabetic patients

	Control	Type I	Type II	Control	Type I	Type II	Control	Type I	Type II	Control	Type I	Type II
	Mg			Mn			Se			Cr		
Mean	19.18	17.64	16.38	13.69	27.67	29.44	6.61	6.32	5.74	4.02	3.52	3.02
±SE	0.27	0.74	0.70	0.19	0.85	0.43	0.10	0.16	0.23	0.16	0.11	0.05
%		-8.04	-14.60		+102.16	+115.09		-4.26	-13.07		-12.36	-24.74
P< D vs C		Ns	0.01		0.01	0.01		Ns	0.01		0.01	0.01
D_1 vs D_2			Ns			Ns			0.05			0.01

Ns: Non significant, Significant (P<0.043), Very highly significant (P<0.001-P<0.0003)

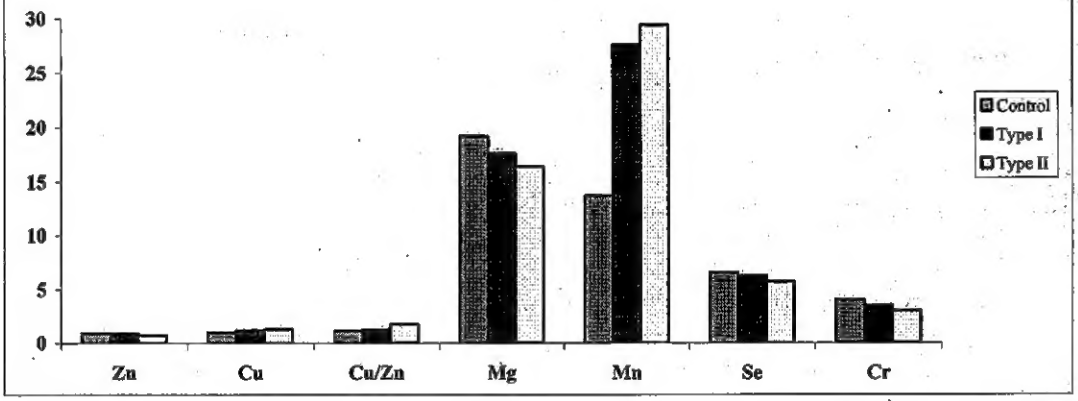
Table (4) Micronutrient status of diabetic patients

	IDDM	NIDDM
Zn	Ns	--↓
Cu	+↑	+↑
Cu/zn	+ns	+↑
Mg	--ns	--↓
Mn	+↑	+↑
Se	--ns	--↓
Cr	--↓	--↓

Table (5): Predictors of effect of diabetes mellitus on trace elements using glucose as the dependent variable.

Variables	R^2	R change	Beta	T-value	P	F test	P <
Mn	0.783		0.874	19.465	0.000		
Mn and wt	0.821	0.038	0.200	4.466	0.000		
Constant (-129.062)						205.517	0.000

Figure (1): Level of trace elements of the diabetic patients



Discussion

Trace elements status (concentration) is altered in the diabetic state, although the factors affecting trace element homeostasis in this condition are not well understood. Diabetes mellitus can disturb the metabolism of zinc, copper, and selenium. The studies of selenium (Se), zinc (Zn) and copper (Cu) levels in diabetic patients have led to contradictory findings as the possible relationship between the degree of diabetic control and the changes in mineral contents (Mertz, 1981; Ruiz *et al.*, 1998).

Zinc (Zn) is an essential element for many enzymes involved in human metabolism and plays a role in the biosynthesis and storage of insulin in β-cells. Insulin is stored in the β-cells as hexameric crystal containing a variable number of zinc molecules. This crystal is released into the portal venous system at the time of β-cells degranulation. Variation of the zn: insulin ratio within this crystal has been shown to alter its antigenic properties (Arquilla *et al.*, 1978 a). Arquilla *et al.* (1978 b) stated that zinc is capable of modulating insulin action, and zn also, enhances hepatic pending of insulin while zn deficiency lead to increase insulin resistance. The mean serum zinc level of type II is decreased when compared to the control one ($P < 0.01$). A decrease in serum Zn may be the result of inadequate dietary

Zn, anorexia, vomiting or various drugs. In addition, a substance known as leukocyte endogenous mediator released from leucocytes during inflammation acts to redistribute the body zinc from the serum to the liver and may produce a drop in serum zn. Zn is mainly bound to albumin, the concentration of which is much more stable. Hypozincema in diabetes mellitus may be due to altered zinc metabolism or tissue deficiency (Garg *et al.*, 1994). The decrease of plasma zinc can reflect a deficient storage or a chronic hyper secretion of insulin in hyperglycemic patients. Kinlaw *et al.* (1983) stated that decrease of Zn might be due to excessive urinary losses uncompensated by increased gastrointestinal absorption. Zinc deficiency has several potential clinical implication as impairement of wound healing, cell-mediated immunity and to cause functional, anatomical and biochemical defects in the retina (Allen *et al.*, 1981; and Leure-dePree, 1982). Mateo *et al.* (1987) think that the quotient zinc/copper might play a role in the pathogenesis of the arteriosclerosis in diabetes. Diabetes mellitus affects zinc homeostasis in various ways, although it is most probably the hyperglycemia rather than any primary lesion related to diabetes which is responsible for the increased urinary loss and decrease in total body zinc (Raz and Havivi, 1989). The decreased concentration of both mg and zn level in type-2 diabetic mellitus probably suggest lower antioxidant status (Anetor *et al.*, 2002). It is known that certain inorganic trace elements such as vanadium, zinc, chromium, copper, iron, potassium, sodium, and nickel play an important role in the maintenance of normoglycemia by activating the beta-cells of the pancreas (Narendhirakannan *et al.*, 2005). Our results for zinc (zn) levels in type i agree with Ruiz *et al.* (1998); Zargar *et al.* (2002). Our results for zn in type II agree with Anetor *et al.*, (2002) and Al-Maroof & Al-Sharbati (2006).

Copper (Cu) is an essential trace element, plays an important role in cytochrome oxidase function at the terminal end of the mitochondria. Plasma Cu is mainly bound to ceruloplasmin, an acute phase protein, which can change owing to a variety of conditions not directly related to copper metabolism. Conflicting results have been reported regarding the Cu level in type I diabetes mellitus, both elevated as well as decreased plasma copper concentrations had been reported (Isbir *et al.*, 1994; and Brun *et al.*, 1992). Fujimoto (1987) stated that copper is not considered to be an important factor in diabetes mellitus due to insignificant change in his study. Our results for cu level in type ii agree with Zargar *et al.* (1998).

Car *et al.* (1992) stated that although the cu/zn ratio was higher in both groups of diabetic (type I & II) patients, it is not related to chronic diabetic complications.

Diabetes mellitus may be associated with magnesium depletion, which in turn may contribute to metabolic complications of diabetes including vascular disease and osteoporosis. Magnesium (Mg) has a potential role in the pathogenesis of disease states such as hypertension and diabetic complications (Resnick *et al.*, 1991 and Grafton *et al.*, 1992). Mg is an important cofactor in numerous intracellular enzymatic reactions including glucose metabolism. Diabetic patients are susceptible to mg depletion and this may be due to glucosuria by causing excessive urinary loss, or due to nutritional factors or hyperinsulinemia (maltezos *et al.*, 2004). Our results for mg level in type i agree with Tuvemo *et al.* (1997); and Zargar *et al.*, (2002). Our results for mg levels in type II agree with Anetor *et al.* (2002).

Insulin or agents that can mimic its action (insulin-mimetics) are necessary to promote the entry of glucose into tissues where the glucose can either be converted into energy or stored for later use. In recent years, selenium has been shown to mediate a number of insulin-like actions both *in vivo* and *in vitro*. These insulin-like actions include stimulating glucose uptake and regulating metabolic processes such as glycolysis, gluconeogenesis, fatty acid synthesis and the pentose phosphate pathway. The mechanism by which selenium is capable of mimicking insulin is not clear; however, reports indicate that selenium does activate key proteins involved in the insulin-signal cascade. Various proteins in the insulin-signal cascade have been shown to be necessary for different insulin-regulated events.

Selenium acts as an insulin-mimetic (Stapleton, 2000). Selenium shows insulin-mimic properties both *in vitro* and *in vivo*. The decreased levels of selenium in sera of diabetic patients (type II) suggest the possible role of glutathione peroxidase activity (Kljai and Runje, 2001). Our results for selenium levels in type I disagree with Ruiz *et al.* (1998). Our results for selenium levels in type II agree with Navarro *et al.* (1999).

Chromium (Cr) is an essential element, contained in gtf (glucose tolerance factor), mainly affects saccharides, it potentiates insulin action via interaction with insulin receptor on the cell surface. The cr level was significantly decreased in diabetic patients compared to controls which agrees with Fujimoto (1987); Zima *et al.* (1998); and Ekmekcioglu *et al.* (2001). Fujimoto (1987) reported that chromium might play an important role in advanced diabetes mellitus. Racek (2003) stated that after absorption of cr in the gastrointestinal tract, Cr is most likely transported to cells bound to the plasma protein called transferrin. Insulin initiates chromium transport into the cells where it is bound to the oligopeptide apochromodulin. This oligopeptide combined with four chromium (III) atoms forms chromodulin, which is important for amplifying the insulin signaling effect. After binding to insulin-activated receptor, chromodulin increases tyrosine kinase activity by one order. This enzyme forms a part of intracellular portion of insulin receptor.

conclusion:

Diabetes can alter copper, zinc, magnesium, manganese, chromium and selenium levels. It is not known whether differences in trace element status are a consequence of diabetes, or alternatively, whether they contribute to the expression of the disease.

References

1. **Allen J, Kay N and Macclain C (1981):** Severe zinc deficiency in humans: association with a reversible t-lymphocyte dysfunction. Ann. Intern. Med., 95: 154-157.
2. **Al-Maroof Ra, And Al-Sharbati Ss. (2006):** Serum zinc levels in diabetic patients and effect of zinc suppl-ementation on glycemic control of type 2 diabetics. Saudi. Med. J., 27(3): 344-350.
3. **Anetor Ji, Senjobi A, Ajose Oa, And Agbedana Eo. (2002):** Decreased serum magnesium and zinc levels: atherogenic implications in type-2 diabetes mellitus in Nigerians. Nutri. Health; 16 (4): 291-300.
4. **Arquila E, Thiene P, Brugman T, *et al.*, (1978 A):** Effect of Zn on the conformation of antigenic determinants of insulin. Biochem. J., 175: 289-297.
5. **Arquila E, Packer S, Tarmas W, *Et Al.*, (1978 B):** The effect of Zn on insulin metabolism. Endocrinology; 103: 1440-1449.
6. **Avram G. (1964):** "Quantitative Data." In: Biostatistics: An Introductory text. Chapter 2, Page 53 & 63. The Macmillan Company, New York, Collier Macmillan Limited, London.
7. **Brun Jf, Fons C, Fussellier M, *Et Al.*, (1992):** Urinary zinc and its relationship with microalbuminuria in type i diab-etics. Biol. Trace Elem. Res., 32: 317-323.
8. **Car N, Car A, Granic M *et al.*, (1992):** Zinc and copper in the serum of diabetic patients.Biol. Treace Elem. Res.,32:325-329.
9. **Chausmer Ab. (1998):** Zinc, Insulin and Diabetes.J Am. College Nutri., 17: 109-114.
10. **Ekmekcioglu C, Prohaska C, Pomazal K, Steffan I, Schernthaner G, And Marktl W. (2001):** Concentrations Of Seven Trace Elements In Different Hematological Matrices In Patients With Type 2 Diabetes As Compared To Healthy Controls. Biol. Trace Elem. Res., 79 (3): 205-219.
11. **Fujimoto S. (1987):** Studies On The Relationships Between Blood Trace Metal Concentrations And The Clinical Status Of Patients With Cerebrovascular Disease, Gastric Cancer And Diabetes Mellitus. Hokkaido Igaku Zasshi., 62 (6): 913-932.
12. **Garg Vk, Gupta R, And Goyal Rk. (1994):** Hypozincemia In Diabetes Mellitus. J. Assoc. Physicians India; 42 (9): 720-721.
13. **Grafton G, Bunce Cm, Sheppard Mc, Brown G, And Baxter Ma. (1992):** Effect of mg^{2+} in etiology of diabetic complications. Diabetes; 41: 35-39.
14. **Isbir T, Tamer L, Taylor A., *Et Al.*, (1994):** Zinc, copper and magnesium status in insulin-dependent diabetes. Diabetes Res., 26: 41-45.
15. **Kinlaw Wb, Levine As, Morley Je, *et al.* (1983):** Abnormal zinc metabolism in type II diabetes mellitus. Amr.J. Med.,75: 273-277.

16. **Kljai K, And Runje R. (2001):** Selenium and glycogen levels in diabetic patients. biol. trace elem. Res; 83 (3): 223-229.
17. **Leure-Depree Ae, And Macclain Cj. (1982):** Effect of severe zinc deficiency on the morphology of the rat retinal pigment epithelium. invest.Ophthalmol.,23:425-434.
18. **Levine As, Mcclain Cj, Handwerger Bs, *Et Al.*, (1983):** Tissue zinc status of genetically diabetic and streptozotocin induced diabetic mice. Am. J. Clin. Nutr., 37: 382-386.
19. **Lioyd B, Holt P, And Delves T. (1982):** Determination Of Selenium In Biological Samples By Hydride Generation And Atomic-Absorption Spectroscopy. Analyst; 107: 927-933.
20. **Maltezos E, Papazoglou D, Exiara T., *et al.*, (2004):** Serum Magnesium Levels In Non-Diabetic Offspring Of Patients With Type 2 Diabetes Mellitus. Diabetes Nutr. Metab., 17 (1) 12-16.
21. **Mateo Mc, Bustamante Jb, And Cantalapiedra Ma. (1978):** Serum, Zinc, Copper And Insulin In Diabetes Mellitus. Biomedicine, 29(2): 56-58.
22. **Melchior T, Simonsen Kw, Johannessen Ac, And Binder C. (1989):** Plasma Zinc Concentration During The First 2 Years After Diagnosis Of Insulin-Dependent Diabetes Mellitus: A Prospective Study. J. Int. Med., **226**: 53-58.
23. **Mertz W. (1981):** The Essential Trace Elements. Science; **213**: 1332.
24. **Narendhirakannan Rt, Subramanian S, And Kandaswamy M. (2005):** Mineral Content Of Some Medicinal Plants Used In The Treatment Of Diabetes Mellitus. Biol. Trace Elem. Res., 103 (2): 109-115.
25. **Navarro-Alarcon M, Lopez-G De La Serrana H. *et al.*, (1999):** Serum and urine selenium concentration as indicators of body status in patients with diabetes mellitus. Sci. Total Environ., 228 (1): 79-85.
26. **Aoac (1984):** "Official method of analysis of the association of official analytical chemists. published by the association of official analytical chemists, Inc., Arlington, Usa.
27. **Paolisso G, Seheen A, D'onofrio F And Lefebvre P (1990):** Magnesium and glucose homeostasis. Diabetologia., 33: 511-514.
28. **Prasad As. (1998):** Zinc deficiency in human: a neglected problem. J. Amr. College Nutri., **17**: 542-543.
29. **Price Wj. (1972):** In: "Analytical atomic absorption spectrometry". heyden son, london.
30. **Racek J. (2003):** Chromium As An Essential Element. Cas Lek Cesk., 142 (6):335-339.
31. **Raz I, And Havivi E. (1989):** Trace elements in blood cells of diabetic subjects. Diabetes Research; 10: 21-24.
32. **Resnick Lm, Gupta Rk, Bhargava Kk, *Et Al.*, (1991):** Cellular ions in hypertension, diabetes and obesity: a nuclear magnetic resonance spectroscopic study. Hypertension, 17: 951-957.
33. **Ruiz C, Alegria A, Barbera R, Farre R, And Lagarda J. (1998):** Selenium, Zinc And Copper In Plasma Of Patients With Type 1 Diabetes Mellitus In Different Metabolic Control States. J. Trace Elem. Med. Biol., 12(2): 91-95.
34. **Stapleton Sr. (2000):** Selenium: An Insulin-Mimetic. Cell Mol. Life Sci., 57 (13-14): 1874-1879.
35. **Steel Rg and Torrie Jh. (1969).** Principles And Procedures Of Statistics. Mcgraw Hill, Book Company, Inc., New York.
36. **The Expert Committee On The Diagnosis Of Diabetes Mellitus. (2000):** Report Of The Expert Committee On The Diagnosis Diabetes Care, 23: S4-S19.
37. **Trinder P. (1969):** Determination of glucose in blood using glucose oxidase with an alternative acceptor. Ann. Clin. Biochem., 6: 24-27.
38. **Tuvemo T, Ewald U, Kobbah M, And Proos La. (1007):** Serum magnesium and protein concentrations during the first five years of insulin-dependent diabetes in children. Acta Paediatr. Suppl.,418 7-10.
39. **Victor Ag, Lielin P De M, And Romer Ar. (1994):** Determination of total chromium in whole blood, blood components, bone, and urine by fast furnace program electrothermal atomization aas and using neither analyte isoformation nor background correction. Anal. Chem., 66: 3624-3631.
40. **Zargar Ah, Shah Na, Massodi Sr, *et al.* (1998):** Copper, zinc and magnesium levels in non-insulin-dependent diabetes mellitus. Postgrad. Med. J., **74**: 665-668.
41. **Zargar Ah, Bashir Mi, Masoodi Sr, Laway Ba, Wani Ai, Khan Ar, And Dar Fa. (2002):** Copper, zinc and magnesium levels in type-1 diabetes mellitus. Saudi Med. J., 23(5): 539-542.
42. **Zima T, Mestek O, Tesar V, Tesarova P, Nemecek K, Zak A, And Zeman M. (1998):** Chromium levels in patients with internal diseases. biochem. Mol. Biol. Int., 46 (2): 365-374.

تأثير مرض البول السكري على تركيز المعادن النادرة في مرضى البول السكري المصريين

غادة زغلول عباس سليمان

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المقدمة: يعتبر مرض البول السكري سبب رئيسي في مضاعفات مرض السكر وهو مصحوب بتغيرات في تركيزات الأملاح النادرة.

الهدف : دراسة تأثير مرض البول السكري على تركيزات المعادن النادرة.

الطرق و المواد: تمت التجربة على 90 شخص موزعين كالتالي: (30 شخص طبيعى ، 30 مصابين بمرض البول السكري من النوع الأول ، 30 مصابين بمرض البول السكري من النوع الثاني) . تم تقدير العناصر الآتية: زنك – نحاس – ماغنسيوم – منجنيز – كروم – سيلينيوم . تم استبعاد الأشخاص الذين لديهم مضاعفات . تم تجميع العينات في أنابيب خاصة بعد فترة الصيام. تم قياس الوزن والطول.

النتيجة : لا يوجد تغيير معنوي في تركيز الزنك ، الماغنسيوم ، السيلينيوم بالنسبة لمرضى النوع الأول. لوحظ وجود ارتفاع معنوي في تركيز النحاس ، المنجنيز بالنسبة لمرضى النوع الأول و الثاني والعكس صحيح بالنسبة للكروم. كما لوحظ وجود انخفاض معنوي في تركيز الزنك – الماغنسيوم – السيلينيوم في مرضى النوع الثاني. وجد أن عنصر المنجنيز أكثر العناصر تأثرا بمرض البول السكري.

الاستنتاج : مرضى البول السكرى يمكن أن يؤثر ويغير من تركيز العناصر مثل : زنك – نحاس – ماغنسيوم – منجنيز – كروم – سيلينيوم .